(FILE 'HOME' ENTERED AT 09:21:10 ON 13 MAR 2000)

FILE 'MEDLINE' ENTERED AT 09:21:20 ON 13 MAR 2000

```
L1         28378 S PROTEIN KINASE C
L2         54819 S ANXIETY
L3             5 S L1 AND L2
L4             0 S ((MESSING RO) OR (MESSING ROBERT O.))/AU
L5            39 S ((MESSING R O) OR (MESSING ROBERT O.))/AU
L6            17 S L1 AND L5
L7         11528 S (TRANSGENIC MOUSE) OR (TRANSGENIC MICE) OR (TRANSGENIC ANIMAL
L8            77 S L1 AND L7
L9             0 S L8 AND L2
L10           17 S L7 AND L2
L11            0 S L10 AND L1
```

FILE 'CAPLUS, USPATFULL, BIOSIS, EMBASE' ENTERED AT 09:29:53 ON 13 MAR 2000

```
L12           94 S L3
L13           65 S L6
L14           40 S L9
L15          149 S L10
L16           40 S L11
L17          209 DUP REM L12 L13 L14 L15 L16 (179 DUPLICATES REMOVED)
L18           90 DUP REM L12 (4 DUPLICATES REMOVED)
L19           36 DUP REM L13 (29 DUPLICATES REMOVED)
L20           40 DUP REM L14 (0 DUPLICATES REMOVED)
L21          125 DUP REM L15 (24 DUPLICATES REMOVED)
L22           40 DUP REM L16 (0 DUPLICATES REMOVED)
L23            1 S PCKE
L24           12 S PKCE
L25            0 S L24 AND L14
```

```
=> logoff hold
```

COST IN U.S. DOLLARS SINCE FILE TOTAL

1 : *Proc Natl Acad Sci U S A* 1995 Apr 25;92(9):3658-62

Related Articles, Books

PubMed Services

Related Resources

Mutant mice lacking the gamma isoform of protein kinase C show decreased behavioral actions of ethanol and altered function of gamma-aminobutyrate type A receptors.

Harris RA, McQuilkin SJ, Paylor R, Abeliovich A, Tonegawa S, Wehner JM

Denver Veterans Affairs Medical Center, CO, USA.

Calcium/phospholipid-dependent protein kinase (protein kinase C, PKC) has been suggested to play a role in the sensitivity of gamma-aminobutyrate type A (GABAA) receptors to ethanol. We tested a line of null mutant mice that lacks the gamma isoform of PKC (PKC gamma) to determine the role of this brain-specific isoenzyme in ethanol sensitivity. We found that the mutation reduced the amount of PKC gamma immunoreactivity in cerebellum to undetectable levels without altering the levels of the alpha, beta I, or beta II isoforms of PKC. The mutant mice display reduced sensitivity to the effects of ethanol on loss of righting reflex and hypothermia but show normal responses to flunitrazepam or pentobarbital. Likewise, GABAA receptor function of isolated brain membranes showed that the mutation abolished the action of ethanol but did not alter actions of flunitrazepam or pentobarbital. These studies show the unique interactions of ethanol with GABAA receptors and suggest protein kinase isoenzymes as possible determinants of genetic differences in response to ethanol.

Comments:

- Comment in: Proc Natl Acad Sci U S A 1995 Apr 25;92(9):3633-5

PMID: 7731960, UI: 95249532

Display Abstract Save Text Order Add to Clipboard

1 : *Shih Yen Sheng Wu Hsueh Pao* 1996 Dec;29(4):429-34

[LPS and PMA induced PKC-alpha and PKC-epsilon activation and translocation in murine peritoneal macrophages].

[Article in Chinese]

Lin MQ, Chang ZL

Shanghai Joint Laboratory of Life Sciences, Institute of Cell Biology, Chinese Academy of Sciences.

Suppressor macrophages induced by the continuous invasion of tumor cells and parasites, which can acquire an ability in vitro to kill or inhibit tumor cells and inhibit the activity of T, B lymphocytes and NK cells, have been indicated. We have developed a procedure previously to modulate the suppressor macrophages by bacterial lipopolysaccharide (LPS). The modulated macrophages remained and even enhanced the ability to inhibit tumor growth and to up-regulate or enhance the activities of T, B lymphocytes and NK cells in vitro. However, the mechanisms of macrophage modulation by LPS are unknown. This investigation was designed to analyze the regulation of PKC activity and to characterize the isoforms of PKC during macrophage modulation by using Western blot and endogenous substrate phosphorylation (PKC-DESP). In rest cells, PKC-beta was found to be the most abundant isoform in macrophages; and PKC-alpha, beta was found predominantly in the cytosol. Using PMA as a positive control, we found that the immuno-modulator agent--LPS triggered the physical translocation from the cytosol onto the membrane of PKC-alpha and PKC-epsilon, but PKC-beta (beta I or beta II) was difficult to detect. The analysis of PKC-DESP showed a pattern with a time course similar to that observed with Western blot. We observed that LPS and PMA increase the level of phosphorylation of 55 kDa and 74 kDa proteins with a corresponding decrease in the cytosolic proteins. It suggests that the translocation of PKC-alpha and PKC-epsilon, may be important events involving in the PKC-pathway by LPS-mediated modulation in suppressor macrophages.